minutes with 10 mM methyl-b-cyclodextrin (MbCD) and another portion was left untreated. Raft and soluble fractions were prepared and analyzed by immunoblotting for GP (upper panel) and for the raft-excluded protein transferrin receptor (TrfR, lower panel).

[0024] FIGS. **2**A and **2**B. Colocalization of filovirus glycoproteins with GMl on intact cells. (A) 293T cells were transfected with the indicated GP, and stained at 4° C. with Alexa488-CTB (green) and anti-GP mAb followed by Alexa-647 conjugated anti-mouse antibodies (red), cells were fixed and imaged using confocal microscopy. Colocalization is represented by yellow appearance in the overlay (right panels). A 3-D reconstruction of the compiled data from 25 sections of a Ebo-GP transfected cell is also shown. (B) 293T cells were concurrently stained at 4° C. with Alexa-488 conjugated anti-TrfR antibody (green) and Rohdamin-CTB (red), fixed and analyzed by confocal microscopy. No colocalization between these two molecules was observed, evident by the lack of yellow appearance.

[0025] FIGS. **3**A, **3**B and **3**C. Localization of filovirus proteins in lipid rafts in infected cells. (A) Primary human monocytes were infected with MBGV. After 24 h cells were lysed in 0.5% triton-XIOO and detergent-soluble (S) and -insoluble (I) fractions were separated by centrifugation, samples were irradiated (2×10^6 R), and analyzed by immunoblotting with a guinea pig anti-MBGV antibody to detect viral proteins NP and VP35/VP40 (lanes 3,4); lanes 1,2: uninfected control; lane 5: inactivated MBGV (1 mg). N. S.: non-specific band. (B) HepG2 hepatocytes were infected with EBOV-Zaire, lysed, irradiated (6×lO R), and rafts (R) and soluble (S) fractions were prepared by ultracentrifugation 24 hours post infection. Ebola GP and VP40 were detected by immunoblotting. (C) Ebola-infected Vero E6 cells were irradiated (4×10^6 R), fixed and stained for Ebola virus (red) and GMl (green) at 4° C. and imaged by confocal microscopy; left panel: single section; right panel: 3D reconstruction of the compiled data.

[0026] FIGS. **4**A and **4**B. Incorporation of GMl in released filovirus virions. (A). Ebola virus was immunoprecipitated from supernatant of infected Vero-E6 cells (lane 2), or uninfected cells as control (lane 1), using anti-GP mAb. After irradiation (2×10^6 R), a fraction of immunoprecipitate (IP) was spotted on nitrocellulose membrane and blotted with HRP-conjugated CTB to detect GMl (lower panel). Another portion of the IP was analyzed by SDS-PAGE and immunoblotting with anti-GP mAb (top panel). (B) MBGV (1 mg), prepared by ultracentrifugation and inactivated by radiation (1×10^7 R), was analyzed for the presence of GMl, TrfR and GP in a similar fashion. As control, rafts and soluble fractions from untransfected 293T cells were used.

[0027] FIGS. **5**A and **5**B. Release of Ebola GP and VP40 as GMl-containing particles. (A) 293T cells were transfected with the indicated plasmids, supernatants were cleared from floating cells by centrifugation and particulate material were pelleted through 30% sucrose by ultracentrifugation. The individual proteins were detected in the cell lysates and in the particulate material from supernatant by immunoblotting (IB). A fraction of cleared supernatant was subjected to immunoprecipitation using anti-GP mAb and analyzed for the presence of GMl (lower panel) as described in the legend to FIG. **1**. (B) The particulate material from cells transfected with GP+VP40 were further purified on a sucrose step gradient and the low density fraction was analyzed for the presence of VP 40 (top panel), TrfR (middle panel), and GMl (lower panel). Rafts and soluble fractions from untransfected 293T cells were used as control.

[0028] FIGS. **6**A, **6**B, and **6**C. Electron microscopic analysis of virus like particles generated by EBOV GP and VP40. Particles obtained by ultracentrifugation of the supernatants of 293T cells transfected with Ebola GP+VP40 were negatively stained with uranyl-acetate to reveal the ultrastructure (A), or stained with anti-Ebo-GP mAb followed by Immunogold rabbit anti mouse Ab (B), and analyzed by electron microscopy. The length of each particle is indicated in mm. (C) 293T cells transfected with Ebola GP+VP40 were immunogold-stained for Ebola GP, fixed, cut, and analyzed by electron microscopy. The picture depicts a typical site of VLP release from the cells, indicated by arrows. A magnification of the site of VLP release is also shown to better visualize the gold staining on the particles.

[0029] FIG. **7**. Inhibition of Ebola infection by raft-disrupting agents filipin and nystatin. Vero E6 cells were left untreated or treated for 30 minutes with 0.2 rag/ml of filipin or 100 U/ml of nystatin at 37° C., washed extensively with PBS and infected with Ebola at an MOI of 1. As a control for lack of general toxicity and persistent effect on viral replication, upon treatment with filipin, cells were washed and incubated in medium for 4 h before infection with EBOV (Filipin (recovered). After 48 h supernatants were harvested and viral titers determined by plaque assay.

[0030] FIGS. **8**A and **8**B. Serum antibody responses in mice following intraperitoneal immunization with 40 ug of EBOV VLPs, inactivated Ebola (iEBOV) or Marburg (iMBGV) virus on days 0, 21, and 42. (A) Total IgG serum anti-Ebola antibodies were measured by ELISA 42 and 63 days post immunization (dpi) following the 2nd or 3rd vaccination, respectively. Ebola antibody titers were measured for individual mice and the results are graphed as the endpoint titer for each mouse. The number of mice with the same endpoint titer are noted on the graph. Closed and filled symbols represent the titer after second and third vaccination respectively. (B) Percent neutralization of Ebola virus infection in VeroE6 cells by sera of immunized mice. Two-fold dilutions of sera were tested for their ability to neutralize Ebola virus infection and are plotted as the mean of the percent neutralization for each group of immune sera as compared to mock-treated VeroE6 cells.

[0031] FIG. **9**. Ebola (e)VLPs protect mice against challenge with mouse-adapted EBOV. Mice were immunized intraperitoneally with 40 ug of eVLPs, iEBOV or iMBGV on 0, 21, and 42 dpi. All mice were challenged on day 63 with 300 pfu of mouse-adapted Ebola virus. Results are plotted as percent survival for each immunization group.

[0032] FIGS. **10**A and **10**B. Marburg virus-like particles (mVLP) are morphologically similar to authentic Marburg virus (MARV) virions. a-b, Electron micrographs of MARV (a) or mVLP (b) at 40,000×. Particles, obtained by ultracentrifugation of the supernatants of MARV GP and VP40 transfected cells or cells infected with MARV virus, were negatively stained with uranyl acetate to reveal the ultrastructure.

[0033] FIGS. **11**A and **11**B. Humoral responses to VLP vaccination. Strain 13 guinea pigs were vaccinated with iMARV (n=5), mVLPs (n=5), eVLPs (n=5) in RIBI adjuvant, or adjuvant only (n=6) three times at three-week intervals. a-b, Serum samples from the guinea pigs were obtained three weeks after the first (1), second (2), or third (3) vaccination and four weeks after challenge (PC). Total serum (a) anti-